

REMOTE STORAGE

816-262-1887L

OAK

Take Library
D.C. and District
Author, Name
Publisher, Name
Call Number, Name
Place, Name
OCLC/Decline

Lending Library
Checkin Date

Staff
Information
Library

GEORGETOWN UNIVERSITY

PUBLIC SERVICES LABORATORY



LEAD POISONING in CHILDREN:

THE PROBLEM IN D.C. AND PREVENTIVE STEPS

Public Services
(Georgetown U.)
A.O. 67803
B-1-1

Public Services Laboratory
Georgetown University
Washington, D.C.

Dr. Selma J. Mushkin
Director

Advisory Group

Frank Bane, Washington, D.C.
Hale Champion, University of Minnesota
John Gronouski, University of Texas
John Haldi, Haldi Associates, Inc.
Richard Musgrave, Harvard University

W. Todd Furniss, American Council on Education
Ralph Huitt, National Association of State
Universities and Land Grant Colleges
Allan Ostar, American Association of State
Colleges and Universities
Robert Pitchell, National University Extension
Association
William Shannon, American Association of Junior
Colleges

Charles Byrley, National Governors' Conference
Brevard Cribfield, Council of State Governments
John Gunther, United States Conference of Mayors
Patrick Healy, National League of Cities
Bernard Hillenbrand, National Association of Counties
Mark Keane, International City Management Association

September 1971

Lead Poisoning in Children:
The Problem in D.C. and Preventive Steps

Selma J. Mushkin Ph.D.
Ralph Freidin M.D.

Public Services Laboratory
Georgetown University
as Secretariat to

District of Columbia Government
Department of Human Resources
Department of Economic Development
Office of Planning and Management

Washington, D.C.
September 1971
Second Printing
April 1972

LIBRARY
UNIVERSITY OF ILLINOIS
AT URBANA-CHAMPAIGN

Explanatory Statement

The statement of the lead poisoning problem is a part of a policy study of lead poisoning in children. The study was initiated in November 1970 under the aegis of the Office of Planning and Management of the District of Columbia and was carried out with the consultation and guidance of an informal interdepartmental task force known as the Ad Hoc Committee on the District of Columbia Lead Poisoning Prevention Case. Members of this Committee included:

David Seidman, Malcolm Hope, Carroll Swanson, Charles Hayman (M.D.) Dudley Anderson, Roslyn Brandon, Marjorie Willcox, Selma Mushkin, and Ralph Freidin (M.D.)

The views expressed and positions taken are not necessarily those of the city government. They are the work of the Public Services Laboratory of Georgetown University that served essentially as secretariat to the Task Force.

The present lead poisoning prevention program in the District of Columbia has many facets, not all of which have been covered by the study made. The principal sets of actions taken by the District of Columbia government, however, are presented below.

In March of 1970 and again in November 1970 the City Council adopted the following measures to prevent lead poisoning and to finance the costs of care of those suffering from lead poisoning.

On March 27, 1970 a new section (2605) was added to the Housing Regulations (Order of the Commissioner No. 70-111) that authorized the securing of specimens of paint, plaster, or structural materials when there are reasonable grounds for believing that a lead hazard exists and the notification of the inhabitants of the lead poisoning hazard. The regulation directs the Director of the Department of Economic Development to order the owner of the building to remove the lead hazard.

On March 30 lead poisoning was made a reportable disease by Order of the Commissioner and as of November 17, 1970 a resolution was adopted (Res. 70-75) that provides (under D.C. Code 32-322) that the financing of health services in cases of lead poisoning in children is in the public interest.

Also, as a model city project, monies were made available for a screening and treatment program in the model city area.

At present (summer 1971) new directions are being sought to carry out more efficiently the District's lead poisoning prevention responsibilities.

Cost of program options and their effectiveness are summarized in *Planning, Programming and Budgeting for City, State and County Objectives, PPB Note 13* to be published by the Public Services Laboratory.

Many thanks are due to Will Keenan for his shepherding of this paper through to publication, to Steffen G. Blendheim for preparing the map included here and to John Steven Robling for his work on the housing census. Each of these men is a Georgetown University undergraduate.

616.862
M97 l

CPA

REMOTE STORAGE

TABLE OF CONTENTS

	Page
Foreword	iv
Some Tentative Observations	v
The Problem of Lead Poisoning in Children	1
Program Options	14
Bibliography	18
Exhibit 1: Guidelines for Sampling the Housing Environment	22
Research Needed	Back Cover

CPA

FOREWORD

An analysis of the problem of lead poisoning in the District of Columbia was undertaken to assess the size of the problem and the various methods of reducing the risks of death, brain damage and other impairments resulting from ingestion of lead in painted surfaces by young children.

The study was initiated as a joint undertaking of the several departments under the general guidance of a task force comprising the Department of Economic Development, the Department of Human Resources and the Office of Planning and Management of the District of Columbia. The Public Services Laboratory of Georgetown University acted as secretariat to the Task Force for the purpose of carrying out the study.

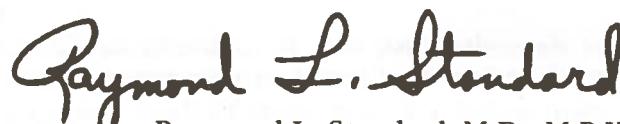
The problem definition statement of that analysis is reported separately for the information of health and housing personnel. We know it will be of great interest to others as well.

The problem of lead poisoning in children has turned out to be both more widespread and more intractable than expected. Lead Poisoning is indeed a preventable disease; means must be taken to inform the public, parents, and health professionals about the nature of the risk.

Dr. Mushkin and Dr. Freidin of the Public Services Laboratory of the Georgetown University have shown that earlier methods of viewing prevalence of lead poisoning started from an erroneous premise, namely, that the lower blood lead levels in children 5-6 years of age could be averaged in to an overall prevalence rate and that for the 5-6 year olds the data suggested low risk. Rather, damage is far more extensive, for the damage may be done to the child in his first few years of life and is no longer apparent in a test for lead in his blood stream at the time he enters school.

Cost of full removal of lead from the 40 percent or so of the houses in the District of Columbia poses a large barrier to full-scale clean up. Partial measures have, in fact, been taken in the District as well as other cities that over a number of years will succeed in making only very little difference. And, a clean up (or de-leading of house units) after the child has suffered lead poisoning is not unlike locking the barn after the horse has been stolen. Preventive action is needed before the child suffers the disease. And that action needs to be carefully tailored to minimize adverse effect on housing supply and rentals. Four possible program alternatives are outlined under "Program Options."

The release of the material that is included in this pamphlet is undertaken to broaden the understanding of the lead poisoning problem among children and to contribute to a better design of steps to prevent the disease.



Raymond L. Standard, M.D., M.P.H.
Director of Public Health
Associate Director, Department of
Human Resources
Washington, D.C.



Julian R. Dugas, Director
Department of Economic Development
Washington, D.C.

SOME TENTATIVE OBSERVATIONS:

1. Extent of the risk.

In the past, the extent of impairment in children attributable to undue body burdens of lead has been understated. Usually prevalence rates are quoted at 10 to 25 percent of the children in the ages under 6 living in core city areas.

These rates underestimate the damaging effects of lead. If brain damage, when done, is irreparable, the higher rates of lead poisoning found in very young children, 1 or 2 years of age, are those that essentially define the minimum extent of the disease. We have viewed the problem of extent of the disease in terms of the risk to children in lead hazard areas of incurring undue body burdens of lead prior to reaching school age. Using data from well baby clinics in the District, we find the risk to be 30 to 50 percent; that is, 30 to 50 percent of babies born in the District of Columbia, who live under lead hazard conditions, may be expected to have an undue body burden of lead sometime before reaching school age, with whatever damaging effects result from such lead burdens. These rates represent the rate of undue body burden of lead found on screening 2-year-olds in well baby clinics, adjusted for those in slum areas who do not come to well baby clinics, and for those who are subject to lead poisoning in later ages.

2. Environmental clean up.

An option often overlooked in prevention programs is the labeling of dwelling units and informing tenants of the risk: "The apartment in which you live may be hazardous to your child's health."

A total clean up of the housing environment is unlikely. The reasons for doubt are (1) the share of all housing units in the older cities that are lead risks (about 40 percent of the units in the District of Columbia); (2) the lack of "fault" on the part of landlords for the leaded surfaces; on the contrary, the more durable and costly paints had lead in them; (3) the extensive period of ingestion of lead prior to lead poisoning that provides the option of parental education—an option that might be supplemented by a public program to remove the hazard in the housing of welfare recipients.

Present practices call for removal of lead hazards (if any are found) from a housing unit after a child has been found to have an undue body burden of lead.

These practices (together with laboratory examination of paint samples) require outlays in the District of Columbia of about \$129 per housing unit in which a cause for deleading is found; the practice on clean up leads to expenditures of \$300 per housing unit (it is reported by landlords or their agents).

Deleading of apartments after a child has been found to have an undue body burden of lead has the following effect: (1) reduces hazard for younger siblings, (2) reduces hazard for repeaters (some small fraction of those with high body lead), (3) reduces hazard of continuing ingestion. But the numbers of children whose risk would be lowered falls far short of the total numbers at risk.

The cost of deleading all the lead risk houses deters such action. House deleading costs might be reduced by technological breakthroughs but it is not very likely that much by way of additional cost saving is possible without significant reductions in labor cost.

What is now known as *facts* about lead damage to children would clearly support a sizeable investment but not the amount required to delead all the housing units in the District.

Total cost of clean up for the estimated 115,000 units in the District is shown below:

Number of Housing Units with Lead:	Cost of clean up (Minimum \$300 per unit)	Likely
115,000 (40 percent of District's total number of housing units)	\$34.5 million	Possibly 3 or 4 times as much
Approx. 15,600- ?2,700 (Leaded housing units in which young children live at one period of time)	\$4-\$7 million	

THE PROBLEM OF LEAD POISONING IN CHILDREN

3. The Cost of Lead Poisoning.

The usual dollar figure cited as the cost of lead poisoning is \$250,000 for each child who becomes mentally retarded.

That figure appears to be computed as a cumulative aggregate over a long period of years for which expenditures would be made on institutional care.

The cost for each year's lead poisoning cases is computed differently here. The cost is shown below for the 120 children who appear during a year at D. C. hospitals with acute symptoms of lead poisoning. Of those 120 children, one-fourth, or 30 children are assumed to suffer brain damage.

The Cost of Lead Poisoning: Each Year Assuming No Action is Taken

Cost in Dollars:

Total:	\$1,047,750
94,500	In-hospital care
600,000	Present value of earnings loss (\$20,000 for each of 30 children)*
353,250	Present value of added education and child development costs (over years up to age 18)**

Cost to Children:

Of each year's births, by the time the children are of school age:

19-80 will be mentally retarded

41-173 will be severely brain damaged

An added 339 to perhaps 1,410 children may suffer some impairment

30-50 percent of those children in lead hazard environments will experience an "undue body burden of lead."

* Assumes alternatively a 6 percent discount rate, no adjustment for trends in earnings or 10 percent discount rate on earnings and 4 percent per annum rise in earnings with a base figure equal to one-half of average wage level for all workers.

** Assumes a 6 percent discount rate or a 10 percent discount rate and 4 percent compensatory rise in prices of public services; extra costs reported at \$1,100 per child.

I. What is the lead poisoning problem?

Lead poisoning in children is a disease resulting principally from the ingestion of lead in lead-based paint or putty. Babies and young children become poisoned with lead by chewing on or ingesting flakes of paint, chips of plaster, and putty from walls which may contain lead. 1-15 About one third of the children in the District of Columbia are exposed to this environmental hazard. Of those exposed, perhaps half have an undue body burden of lead some time before they reach school age.

Children living in housing units that contain loose paint and plaster, and who receive relatively little parental affection and supervision, have much higher risks of lead poisoning than children who are more carefully supervised or live in houses where the paint and plaster are tight, or in newer housing units. 10, 12, 14-19

Between 6 months and 2 years of age, most children mouth objects as they pass through two stages of normal development, which have special importance in understanding the epidemiology of lead poisoning. Most children mouth objects in the middle of the first year as part of their hand-to-mouth exploration, or oral stage, which usually is completed by 18 months. At about 6 months, the children start teething. This stage overlaps the former by lasting until the child is about 2 years old. 20

During the first two years children may consume anything that is loose which they find in their playpen or on the floor. Sometimes they "work" at loosening the paint and plaster near their crib or where they play. While teething, children chew particularly on hard surfaces such as window sills and frames, door frames, stair treads, and furniture. Thus, between 6 and 24 months or so, in the process of normal development, a child may accidentally poison himself with lead if it is accessible to him in his environment. 12

In some cases the oral stage persists as a major activity beyond 18-24 months; 21 and some children develop pica—a compulsion to consume a particular non-food substance. 2, 5, 7, 8, 10, 12, 19, 22 It has been suggested that pica occurs as a result of some imbalance in the maternal-child relationship. 21 Those children who complete the hand-to-mouth development at a very early age have a shorter time of potential exposure to lead hazards.

In the study of 859 children in the District of Columbia, pica was observed to be a symptom chiefly of preschool children. The greatest incidence was found in children between 18 and 24 months of age. The incidence was also found to vary by socioeconomic status. The incidence was between 50 and 60 percent in the 18-24 month age group from low socioeconomic status, while it was about 30 percent in higher socioeconomic groups. After 2 years of age, the incidence dropped; and by 3 to 4 years of age, the habit was almost entirely gone in higher economic groups but remained at about 20 percent in lower economic strata. 23 Pica does not usually occur in children beyond 6 years of age, with the exception of patients with brain damage or schizophrenia. 21 These findings help explain the high prevalence of lead poisoning in lower socioeconomic children between 18 and 36 months, and its confinement to those less than 6 years of age.

II. Special characteristics of the lead poisoning problem.

- (1) Lead is a non-essential element found in trace amounts in all humans. 14, 24
- (2) Adults as well as children have a daily intake of lead from the food, water, and air. 12, 14, 25-29 City dwellers have a higher daily "dosage" of lead than those living in rural areas. 30, 31
- (3) The body is capable of excreting the daily lead intake except when it becomes "excessive."* The time period over which a child eats lead and the amount he consumes determines whether the dose is "excessive." The exact quantity of leaded materials and rate of consumption necessary to ingest an "excessive"

* Excessive is used to mean a daily lead intake greater than the total amount of lead the body is capable of excreting per day.

amount of lead is not known. As long as the "excessive" ingestion continues there is progressive accumulation of lead in the body. 12, 14, 25, 30, 32-34

- (4) It is reported that about 90 percent of the lead ingested orally is unabsorbed; the remaining 10 percent is absorbed into the blood stream and deposited in bone and soft tissues such as brain, lungs, liver, spleen, and bone marrow. Of the lead entering the body by breathing, depending upon the amount of lead intake, between 15 and 20 percent is absorbed into the blood and distributed to bone and soft tissues; the remaining is expelled. 10, 14, 23, 26, 32, 34-38
- (5) One investigator estimates that it takes twice as long for the body to release the lead from the bone and soft tissue as it took to deposit there. 25, 32
- (6) The lead, which is absorbed and deposited in the child's tissues, will become toxic enough to produce *acute encephalopathy** after continuous excessive intake over a period of at least three months. 12, 42 The exposure (length of time, and amount of lead) required to elevate the blood lead level above "normal" is not known. However, it must be less than 3 months and more likely is in the range of 3 to 6 weeks with the length of time varying inversely with the dosage or amount of lead ingested. 25, 42, 43 Dr. J. Julian Chisolm wrote on this question of length of exposure: "It is probable that a minimal period of three months of exposure results in lead poisoning due to ingestion of lead paints." 51
- (7) Young children, under 2 years of age, are especially susceptible to the effects of absorbed lead because they ingest excessive quantities of lead at a time when the cells of their nervous system and other organs are still developing and vulnerable. 11, 42, 44-48
- (8) It is not known what the exact pathological consequences are of an "undue absorption" of lead in a child without clinical symptoms. However, several physicians believe that most children with abnormal blood lead levels demonstrate learning problems, behavioral difficulties, lack of sensory perception, and psychological consequences. 10, 22, 48, 49
- (9) If a child is treated for an excessive body burden of lead and is then re-exposed, he has a significantly "increased risk of having severe neurological damage (retardation, blindness, seizures, or behavioral disorders). 10, 12
- (10) The majority of children with blood lead levels greater than 80 micrograms per 100 milliliters of whole blood are without symptoms. 8, 50 However, as Dr. Chisolm wrote: "Clearcut symptomatic cases of lead poisoning are almost always associated with blood lead concentrations greater than 80 micrograms; thus concentrations of this level should be regarded as high risk levels." 51
- (11) If lead intoxication is not recognized when there are vague symptoms, the child will continue to ingest lead and incur a high risk of developing acute lead encephalopathy. 12, 13, 27, 42, 51
- (12) Children who have undue absorption of lead may precipitously develop acute lead encephalopathy with the onset of any infectious disease or the seasonal change from spring to summer. 13, 27, 42
- (13) Significant improvement in therapy for acute lead encephalopathy should reduce mortality to less than 5 percent of the children. Previously the death rate was substantially higher. 10, 18, 52-55

* *Acute encephalopathy*—Lead intoxication usually has an insidious onset with vague symptoms: listlessness, loss of appetite, constipation, abdominal pain. Occasionally, these symptoms will develop rapidly and be associated with central nervous system involvement: drowsiness, headache, loss of balance, convulsive seizures, coma, and even respiratory arrest.

Table 1: Effects of Excessive Lead Ingestion in Children 9 Months up to 6 Years of Age*

	Rates per 1000 children subject to lead hazard				
	Total	Deaths	Mental retardation	Other severe brain damage	Other brain damage
Acute episode	<6	<<1	2	2	<2
80 micrograms or more on initial screening	14	(See above)	2	3	Unknown
50-79 micrograms	90		1	4	Unknown
40-49 micrograms	140		1	4	Unknown
Total	250	<<1	6	13	Unknown

* Rates of lead burdens are based on New York City data. Michael J. Specter and Vincent F. Guinee, "Epidemiology of Lead Poisoning in New York City—1970," presented at the American Public Health Association's 98th Annual Meeting, Houston, Texas; October 26, 1970 (processed).

Rates of death, mental retardation, etc.—medical journal article findings (see bibliography).

III. Undue absorption of lead, or the defining of lead poisoning.

The definition of the disease process caused by undue absorption of lead (in common with problems of defining many other disease conditions) requires:

- (a) the application of some standardized measures to differentiate the range of "disease" conditions from a range of the "normal."
- (b) the availability of diagnostic tests capable of differentiating the normal from the diseased — tests which can be administered, examined and interpreted by different observers with small margins of error.

(1) The District of Columbia has adopted a standard of undue lead absorption at a level in excess of 40 micrograms of lead per 100 milliliters of whole blood. [This standard is set forth as a recommendation in a 1970 report of the Surgeon General of the Public Health Service. 6]

(2) The average blood lead levels occurring in samples of normal children and adults without excessive exposure range between 13 and 25 micrograms per 100 milliliters of whole blood. 15, 28, 30, 31, 56, 57 In a large study of "normal" blood lead levels in urban dwellers, the mean lead level of 585 specimens obtained from 16 different countries was 17 and the standard deviation was 9. 31 In the three urban areas sampled in the United States the mean lead level values were 17, 18, and 19. 31 Therefore, a standard of 40 micrograms is more than two standard deviations greater than observed means and hence false positives are not likely.

(3) Administration of a test for lead requires that a blood specimen be collected. A blood lead value, however, is only a limited indicator of the body burden of lead. 2, 58 Blood lead levels do not reflect the large fraction of the total body burden deposited in bone. Episodes of high soft tissue concentrations of lead which may have occurred in the

past are not reflected; rather recent and current level of exposure and absorption are. The blood lead test thus has been characterized as measuring the "tip of an iceberg."

(4) Direct analysis of bone or soft tissues for excessive body lead, though possible, is not clinically feasible. Even urine samples are not appropriate for screening purposes according to Dr. J. Julian Chisolm. Blood sampling, as the sampling of the most accessible tissue, is being used in screening programs to indicate the body burden of lead. 25, 33, 44

(5) The blood test for lead is essentially a way to screen for current exposure to lead. It may have to be repeated at fairly frequent intervals (every 8-12 weeks) because large increases in blood lead levels can occur within a short period. 6, 33, 51 A two- or three-fold increase in blood lead level may occur within 2 to 6 months of continued undue absorption once the patient has accumulated an increased body burden of lead. 8, 12, 13, 51 This increase can move the blood lead level from below the normal range (less than 40 micrograms) to substantially above it to the range where acute encephalopathy is a significant risk.

(6) Measurement of whole blood lead requires careful interpretation because it is affected by many factors. Almost all of the blood lead is carried on the surface of red blood cells; blood lead values must be corrected for any wide deviation from the normal amount of blood cells (hematocrit). This is particularly important since the children with high risk of lead poisoning also have a high risk of anemia. 6, 13, 19, 27, 33, 42 Blood lead values are also influenced by the rate of absorption, duration of absorption, and rate of deposits and release of lead from the bone tissue. 33, 36 Furthermore, for some unknown reason, blood lead levels have significant seasonal variation, being increased during summer months. This is reflected by more cases of lead poisoning and acute lead encephalopathy reported during the summer. 1, 2, 7, 10-12, 18, 42, 59 Children who have an undue body burden of lead, but have been without symptoms during the winter, may suddenly develop lead encephalopathy during the summer. 2, 12, 58

(7) Again, children with excessive body lead do not necessarily have symptoms of lead poisoning. Observable disease usually does not occur until the blood lead level is greater than 80 micrograms per 100 milliliters of whole blood; and even then, in less than half the cases. 13, 33, 50, 58

Table 2: Large Numbers of Children With Lead Poisoning Have No Symptoms

One Study Shows:

Blood Lead Levels	Percent with No Symptoms and Positive Urine Test for Lead
60-79 micrograms lead per 100 milliliters whole blood	66%
80-149 micrograms	53%
150+ micrograms	6%

Source: Greengard, J., et al.: Medical Progress in the Prevention of Childhood lead Intoxication, *Illinois Medical Journal*, May 1968, 615-618

IV. Application of standard of undue lead absorption.

The District of Columbia has accepted a verified* analysis of 40 micrograms lead per 100 milliliters whole blood as the criterion for filing a complaint with building inspectors, leading under present housing regulations, to an examination of the housing unit in which the child with an undue body burden of lead lived.

For (a) referral of children for additional diagnosis, and (b) treatment of the child where clinically indicated, a criterion of 50 micrograms lead per 100 milliliters of whole blood has been accepted.

Physicians and hospitals are required by District ordinance to notify the health department of a case of lead poisoning when a diagnosis is made. This notification also sets in motion a housing inspection if, earlier, a complaint was not filed.

The level of 40 micrograms is *not* generally accepted as standard for treatment of the child with chelating agents.** However, depending on the results of additional tests and the severity of symptoms, some of these children may require such therapy. According to the Surgeon General's report, the use of chelating agents is clearly indicated in children who have a blood lead level of 80 micrograms or more. Those children whose blood lead level is between 50 and 79 micrograms require additional testing to determine whether chelating therapy is indicated. 6 One expert suggests that 1 out of 4 children with blood levels of 40 micrograms, or "undue lead absorption," requires chelation. 59

V. The size of the population at risk and of the environmental hazard in the District of Columbia.

The estimated number of children in the District of Columbia *9 months up to 6 years of age*—the ages of major risk—is 62,547. 60 (This figure is a combined total of 49,851 computed from data reported on children under 5 from the 1970 Census of Population First Count Tape (Table 18) adjusted to exclude those below 9 months of age based on data on live births in the District for 1969 and 1970. To this estimated number is added the 5-year-olds—12,696—making the total of 62,547.)

Not all children in these young age groups are uniformly subject to the hazard. *The "hazard" is a result of three factors:*

- the lead content of the paint (or putty) available to the child in his living quarters, or where he spends a considerable part of his time such as day care centers or homes of baby sitters,
- the condition of the painted surface (flaking, etc.), and
- conditions under which the child is allowed to ingest paint, e.g., pica, or lack of parental knowledge about lead risks for children.

How many children are at risk is not a simple question to answer. There are no data on the number of lead-painted housing units in which children live or spend considerable time, and no data on the number of children in housing units in which lead paint is flaked and constitutes a large hazard; nor do we have direct data that would suggest the magnitude of the problem of lack of parental knowledge about the lead hazard for their young children.

Use of lead paint on interior surfaces has been banned but lead paint as an exterior paint is still widely used.

(a) The 1970 Census reports 278,444 occupied housing units in the District of Columbia. Based on earlier Census data and housing demolition and conversion, it is estimated that over 40 percent (or 115,000) of the housing units were built prior to

* Two blood lead tests of 40 micrograms or more.

**Chelating agents: chemical compounds whose molecular structures enable them to bind and form stable compounds with metals such as calcium, mercury, gold, silver, and lead.

World War II when lead paint was widely used for interior surfaces. (This estimate is based on the Census of Housing number of housing units in existence in 1940—185,128—adjusted to exclude 20,000 demolitions and other losses in each of the three decades or so. The data are for the 1950's; additionally, a similar number of demolitions and losses is assumed in the 1940's and 1960's, or a total of 60,000 units. Mergers and conversions on a net basis in the 1950's, again the only period for which such housing change data are available, point to mergers in excess of conversions. It is assumed that possibly as many as an additional 6,000 units in the approximately 30 years may have been so renovated as to no longer constitute a lead hazard despite the construction of the units prior to World War II. With the added 6,000 units, a total of 66,000 units are assumed to have been removed or remodeled of the 185,000 in existence in 1940, leaving 115,000 to 120,000 as hazards.)

Housing units built after World War II have been found to have lead-painted interior surfaces. In the District of Columbia lead has been found on inspection in relatively newly built units. The assumption is that the tenant repainted, and used exterior lead-based paint in doing so.

How many housing units with leaded paint (or putty) are in a state of disrepair where the surfaces may more readily be accessible to young children? Not all of the more than 115,000 housing units built prior to World War II are in a deteriorating condition that would suggest paint flaking and other major lead surface hazards to children. The 1960 Census of Housing contained data on the dilapidated and deteriorating housing. Use of these data as a measure of housing hazards has been suggested by the Bureau of Community Environmental Management of the U. S. Department of Health, Education, and Welfare. In 1960, some 3,200 dwelling units in the District were dilapidated and an additional almost 20,000 were deteriorating, making a total of 23,200—a number perhaps 10 percent of the 1960 total number of dwelling units. If one takes the standard of dilapidated and deteriorating housing as indicative of an environmental hazard, and applies the 10 percent to the total housing units in the District, some 28,000 are lead hazards. Not all of these, however, are inhabited by young children, and even a smaller number are inhabited by young children whose supervision is not an adequate safeguard.

The 62,547 young children (9 months up to 6 years of age) estimated for the District live in a smaller number of dwelling units—perhaps 56,860. (This number would be computed if it were assumed that with multiple young children in one family unit, there are about 1.1 young children per dwelling unit; at 1.6 young children per unit the number of units would be 39,092.) Further, of the 56,860 (39,092) or so units in which young children may live, perhaps 60 percent are non-leaded dwelling units, and 40 percent lead-painted units. (The 60-40 proportions assume young child tenants are distributed among lead-painted and non-lead-painted units in the same proportion as total lead or non-lead units exist in the District.) Thus, perhaps 40 percent of the units, or 15,600-22,700 would be leaded, and 60 percent, or the remaining number, non-leaded. Young children may be found, however, in somewhat larger proportion in public non-leaded housing built in the last decade or so.

But there tends to be considerable movement of families from one housing unit to another, particularly in the core of cities; moves increase the number of children exposed during the course of a year and even a larger number exposed during the course of their first years of life.

We estimate that 25,000 young children (9 months up to 6 years of age) live at any one time during a year in housing units with leaded surfaces or other leaded paint. If only 20 percent of those children were free of pica and had parents who understood the hazards of lead to health, some 20,000 young children would be at risk. Clearly there is a wide margin of error in this number, but again, the limits are the total number of young children, or roughly 62,500, at the one extreme, and all or some fraction of the 20,000 at the other.

A series of data questions arise in establishing the extent of the hazard more precisely, and the cost of the environmental cleanup. These questions may be enumerated

as follows:

(1) How many of the lead-painted dwelling units are inhabited at any one time by families with young children?

Census data of 1970 that are now becoming available will provide the data on distribution of young children among families, and will permit the showing of the average number of young children per family and the distribution of families and households by numbers of children, and perhaps by age of dwelling unit.

(2) How many housing units are inhabited by young children over a period of years? What is the turnover among families with young children? Are dwelling units of a given size and in a specific site rented by families with different (or the same) size, and age of family members?

What is essentially being sought is information that could provide material on the impact of a household cleanup on the actual hazard to young children. If a housing unit is made lead "safe," will this safeguard young children? Or, do many families change their residence with sufficient frequency that the cleanup in their specific dwelling units will not necessarily reduce the risk?

Data were not available at the time this study was being carried out that would provide a basis for adjusting the information to show tenant turnover.

(3) Are there many housing vacancies in the District so that a deleading cleanup may go forward without raising rentals? Or is there a shortage of housing units?

For example, the 1970 housing census shows an overall vacancy rate for rented properties of 5.6 percent. 60

VI. Lead Poisoning: Rates in the District of Columbia.

In the week of June 2-8, 1970, a lead screening program on a citywide basis was carried on in the District of Columbia. The mean blood level for all children screened (826 blood specimens) was 25 micrograms lead per 100 milliliters of whole blood. The mean blood lead level for all those under 5 years of age was 26.4 micrograms. 63 These values are very similar to what has previously been reported from a random group of urban children. 15, 26 However, they are significantly greater than international 31 and national 56 means of "normal" blood lead levels.

Of the children screened in June 1970 in the District of Columbia, 8.6 percent of those 5 years of age or under had lead levels equal to, or greater than, 40 micrograms. In the core areas of the city, a higher percentage, 11.6 percent, were found to have an undue body burden of lead. 63 No child, among the 476 screened 5 years of age or under, had a blood lead level of 60 micrograms—the standard for defining a case of lead poisoning in New York City. 1

A model city screening program was initiated in the District during the months in which this report was underway. In 4 weeks, during May 1971, the findings according to the District officials were similar to those found in the 1970 screening program, or about 10 percent of those tested were found to have 40 micrograms blood lead levels or more. However, the total number of children tested during the first 4 weeks was only about 270 out of the 11,000 children estimated by the health department to live in the model city area, and there was considerable evidence that children most likely to be at risk were not among those tested.

Findings derived from the lead screening in well-baby clinics in the District of Columbia (October 1970 to March 26, 1971) are as follows:

(1) 22 percent of the children 24 to 36 months of age using well-baby clinics have blood lead levels of 40 micrograms per 100 milliliters, or more.

(2) About 26 percent of the children 24 to 36 months of age in lead hazard areas of the District have 40 micrograms lead per 100 milliliters whole blood when an approximate correction is made for non-participation of poorer children with

larger risks. [Chicago data 61 were used in estimating the correction: that is, those data were used to ask: What changes would have occurred in the average rate if children from all economic areas had participated in the screening to the same extent?]

(3) About 31.4 percent of the 2-year-old children have excessive lead if the highest rates (clinic #2) are used as a base. This figure is still too low for 2-year-olds in that neighborhood because there is substantial nonparticipation of the poorest and possibly highest risk children. The adjusted number, again based on Chicago data would be 37 percent.

Much attention has been given in the past to average prevalence of lead burdens in the body among children.

It has been cited for example that 25 percent of those 6 years of age or under have 40 micrograms lead per 100 milliliters whole blood in blood specimens. Such averages are misleading. A new approach is indicated in the way in which the size of the problem is defined. It is an approach that reflects both the high prevalence of excessive body lead among very young children and the irreversibility of much of the brain cell damage. The risk is essentially a peak figure rather than an average figure.

In addition to the adjustment of rates of undue body burdens of lead for nonparticipation among those 2 years old the data also have to be corrected for seasonality and, if possible, for extent of lead hazard in the home.

The basic well-baby clinic data for 2 year olds, now available, are for the winter months. A seasonal adjustment would bring the 26 percent average annual figure almost to 45 percent, since the rate of undue body lead in the peak month of June rises to over 1.7 times the yearly average blood lead levels [Seasonal computed from Chicago screening data 62] and the 37 percent would become when seasonally adjusted, 63 percent.

A piecing together of the District body lead prevalence rates and data from New York City and Chicago suggests the following:

(1) Upwards of 10 percent of the young children under 6 years of age in the lead hazard areas of the District have undue body lead. (The 10 percent is clearly a minimal number and is not a good yardstick of the damaging effects of lead.)

(2) The 22 percent reported for blood lead levels greater than 40 micrograms by well-baby clinics for those 24 to 36 months old using the clinics is the equivalent of 26 percent when minimally adjusted for nonparticipation of those especially at risk and to perhaps almost 45 percent of the young children in lead hazard environments in the peak months.

(3) In some areas of the city the rate of occurrence of undue body burden of lead is even higher.

VII. Regions of concentration.

The high blood levels may be expected to be concentrated in the older, once fashionable, residential areas where expensive lead paint was used. For Washington, D.C., this means the area between Massachusetts and Florida Avenues on the west of New York Avenue, and in the areas around Columbia Road, Harvard Street, and Georgia Avenue. Few cases of high blood levels would be expected to be found in public housing constructed after World War II and in the areas where such public housing is concentrated, namely in Southeast Washington below Alabama Avenue and Wheeler Road. 63 However, cases have been reported from the Southeast across the Anacostia River, and from public housing units. A map of housing units in which lead was found after a complaint was filed shows a wide distribution throughout the city, but particularly east of Rock Creek Park.

VIII. Housing regulations.

By Order of the Commissioner No. 70-111, housing regulations were amended and

Leaded housing of first 700 inspections in reported lead poisoning cases in the District of Columbia



procedures were set up by which the Accident Prevention Division in the Department of Human Resources reports to the Department of Economic Development the finding of an undue body lead in a child. This report is taken as reasonable grounds for believing that "a hazard exists to the health of one or more of the inhabitants . . . because of the presence of lead or its compounds in the paint, plaster, or structural materials of any interior surface," in the words of the Commissioner's Order 70-111.

The regulations issued under the Commissioner's Order 70-111 (amending Section 1102 and to insert 2605.2 and 2605.3) relative to lead paint on exposed interior surfaces of dwellings place responsibility on the Department of Economic Development for securing specimens of paint, plaster, or structural materials when, upon inspection, there is a finding of the presence of flaking, peeling, chipped, or loose paint that may constitute a hazard, or that there are other reasonable grounds for believing there to be a health hazard. If said analysis reveals the presence of lead or its compounds in a quantity in excess of 1 percent by weight, or in a quantity otherwise sufficient to constitute a hazard to health, the Director of the Department of Economic Development is required to notify the inhabitants of the hazard, and is required also to order the owner of the building either (1) to cease occupying, or (2) to remove materials containing lead or its compounds (or otherwise to cover such interior surfaces that constitute a hazard).

Guidelines for environmental sampling are attached as Exhibit 1.

New X-ray fluorescence detectors measure lead in terms of micrograms of lead per square centimeter of exposed surface and at this time the District of Columbia is moving to use such detectors in place of older gravimetric methods of analyzing paint for lead that used the 1 percent by weight standard.

In enforcing the code it is clear that the regulations could be used to require a sampling of the quantity of lead in over 40 percent of the houses in the District, given their age; and if lead in sufficient quantity exists to constitute a hazard, the ordering of owners to cease occupying, or to eliminate such lead paint hazard.

IX. Consequences for the child with lead poisoning.

The consequences of excessive prolonged ingestion of lead are death, mental retardation, other severe brain damage, as well as other behavioral and possibly physical impairments.

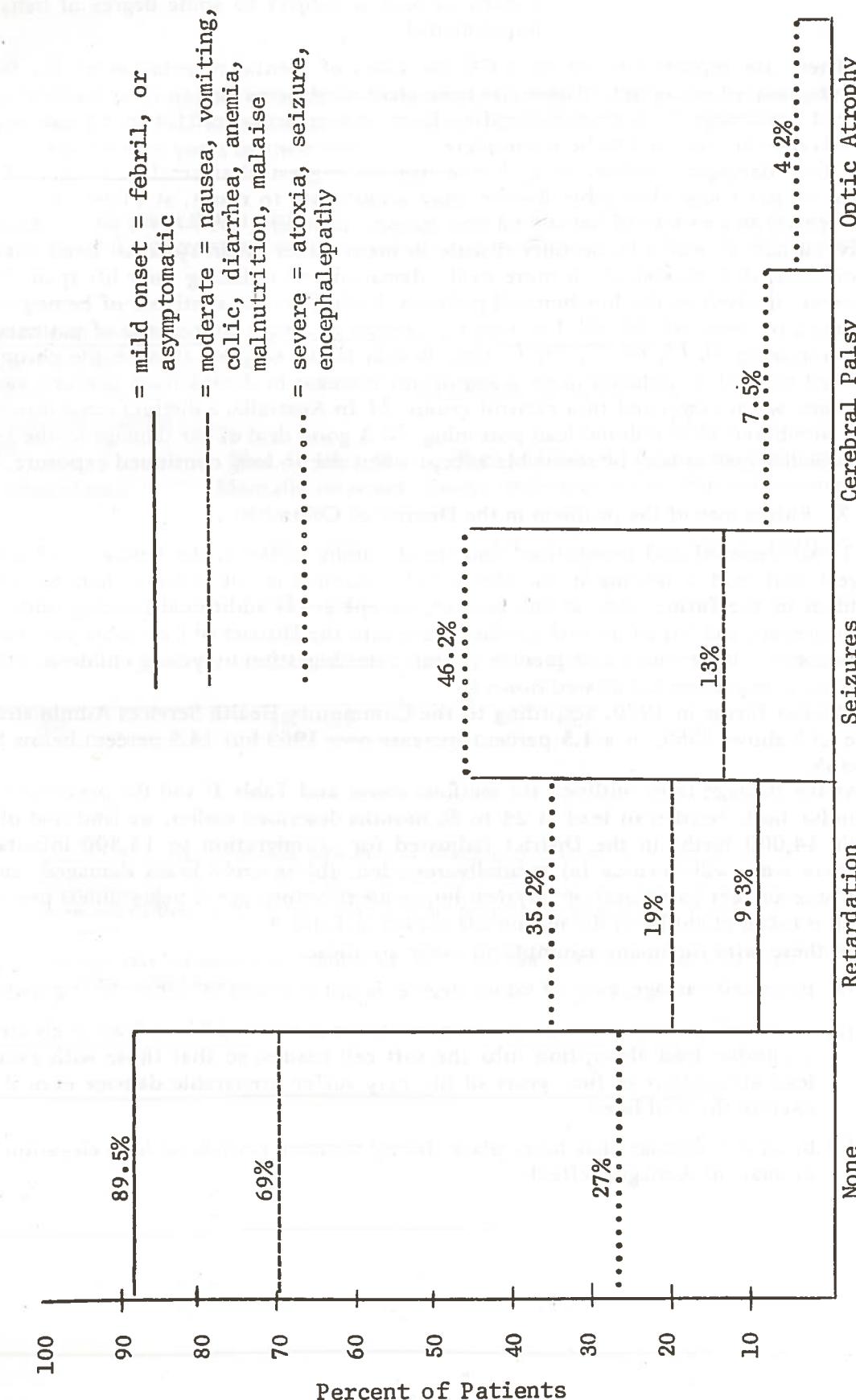
Deaths: Severe lead poisoning can result in death. Recently, with the use of British anti-lewisite (BAL) and ethylenediamine-tetracetate (EDTA) in combination, the fatality rate has been reduced to about 5 percent of acute lead encephalopathy cases.^{5, 10, 13, 54, 55} One known lead poisoning death occurred in 1970 in the District of Columbia. The vital statistics office reports no recorded deaths for the period 1965 through 1969. However, it must be recognized that lead poisoning deaths may not have been identified by the physician since there are no characteristic symptoms which are indicative of the disease as well as the physicians' general lack of awareness of the seriousness and prevalence of this disease.

Mental retardation, other severe brain damage, and other behavioral damage: Lead affects brain cells and causes a variety of different types of damage to the nervous system such as mental retardation, or other brain damage that includes seizures, cerebral palsy, and optic atrophy.^{49, 65, 66}

Tentatively based on findings reported in several medical journals, the following rates (see table 1) are considered reasonable measures of brain cell damage caused by lead ingestion:

Mental retardation:	6 per 1,000 children 9 months up to 6 years of age living under lead risk arrangements (or 2.4 percent of children with undue body burdens of lead)
Severe brain damage other than mental retardation:	13 per 1,000 living under lead risk arrangements (or 5.2 percent of children with undue body burdens of lead)

Table 3: Sequelae of Plumbism in 425 Patients Who Survived



Other behavioral damage (Speech, or other disorders): Not known. (We assume for purposes of question raising that perhaps 1 out of each 2 children with undue body burden of lead is subject to some degree of behavioral impairment.)

There are reported to be in 1970 six cases of mental retardation in the District Training School whose retardation has been attributed, according to their medical record, to lead poisoning.⁶⁷ Unless a specific effort was made to test for lead poisoning, the records may be expected to be incomplete.

Other damage: Further, several investigators suggest that small amounts of lead, which do not cause observable disease, may accumulate to result, at a later age (e.g., age 8-10 years) in a variety of behavioral and speech disorders.^{17, 33, 46, 48} Excessive body burden of lead can produce disease in many other organ systems. Lead coats red blood cells, thus making them more easily damaged and reducing their life span.⁶⁸ The enzymes involved in the biochemical pathway leading to the synthesis of hemoglobin is inhibited by lead.^{46, 69, 70} The result is anemia in a high percentage of patients with lead poisoning.^{6, 13, 19, 27, 33, 42} One British study suggests that people chronically exposed to lead in industry have a significant increase in deaths from cerebral vascular accidents when compared to a control group.⁷¹ In Australia, a distinct renal disease has been attributed to childhood lead poisoning.⁷² A good deal of the damage to the kidney and vascular system may be reversible except when due to long continued exposure.

X. Future size of the problem in the District of Columbia.

Urban renewal and renovation, and the declining births in the District of Columbia suggest that lead poisoning in the District of Columbia is not likely to become a larger problem in the future than at the present, except as (a) additional housing units come into disrepair, and (b) additional families move into the District of Columbia who have no awareness of the serious consequences of lead paint ingestion by young children. (And, at this time immigration has slowed down.)

District births in 1970, according to the Community Health Services Administration, were 215 above 1969, or a 1.5 percent increase over 1969 but 14.9 percent below births in 1965.

At the damage rates outlined (in sections above and Table I) and the prevalence rates of undue body burden of lead at 24 to 36 months described earlier, we find that of each year's 14,000 births in the District (adjusted for outmigration to 13,300 infants) the numbers who will become (a) mentally retarded, (b) severely brain damaged, and (c) otherwise subject to mental or physical impairment before age 6 years unless preventive action is taken might be in the magnitude shown in Table 4.

In these rates the major assumptions made are these:

- (1) brain cell damage, even of minor degree, is not repaired by subsequent growth.
- (2) brain cell damage takes place whenever there is elevated blood lead levels creating an undue lead absorption into the soft cell tissues, so that those with excessive lead absorption in first years of life may suffer irreparable damage even if they excrete the lead later.
- (3) brain cell damage that takes place during summer periods of lead elevation has a permanent damaging effect.

Table 4: Estimated number of children who would be impaired as a consequence of being exposed to lead hazard in home or other living place during the first six years of their lives.

(Assuming 14,000 resident births each year in the District of Columbia, usual outmigration, and several different assumptions about the occurrence of undue body burden of lead.)

Assumed percentages of children whose homes and other living places are subject to lead paint hazard. (In percentages)	ILLUSTRATIONS I AND II					
	Illus. I	Illus. II	Illus. I	Illus. II	Other mental damage	Severe brain damage or physical impairment
50	48 - 80		104 - 173		846 - 1410	
40	38 - 64		83 - 138		677 - 1128	
30	29 - 48		62 - 104		522 - 846	
20	19 - 32		41 - 69		339 - 564	

*Illustration I uses estimated 26% rate of undue lead absorption adjusted upward to 30 percent to reflect exposure in years beyond the second year of life. Illustration II is computed on the basis of 45 percent rate as seasonally adjusted, increased further to reflect exposure in years beyond the second year of age. (50 percent).

**Damage rate includes that counted earlier as mentally retarded or severely brain damaged. (See Table 1).

Program Options

Actors:

The major "actors" involved in lead poisoning are (1) the child and (2) the lead content in the paint of his living quarters. Still additional actors include (3) the parent and/or other babysitters, (4) the landlord (and rental agent) who has certain liabilities for the condition of the housing unit and (5) the public agencies. In the case of diagnosis and treatment, and to prevent further deterioration in the child, the several agencies involved include the Departments of Human Resources and Economic Development and also (6) providers of health care—the practitioner, hospital, well-baby clinic, etc.

For each of the major behavioral units, or actors, the question being asked is: What could feasibly be done about altering behavior or condition of the child, and his living arrangements? For example, health education can be the way to change the behavior of the parent and perhaps even the small child. (The "pain" of medical care possibly can be used as a conditioner against chewing of paint surfaces by the child.)

Housing units can be changed prior to occurrence of a risk or after, and by removal of 1) the access of the child to lead paint, or 2) removal of (a) all lead paint, (b) flaking paint, (c) flaking paint in the room or rooms used most frequently by small children in the household, (d) flaked paint plus chewable surfaces within reach of the child in the room or rooms used by the child most frequently, and so forth. The housing market condition may be changed by better consumer information about lead hazards.

Two Sets of Time Dimensions:

For the Children:

(a) backlog of lead ingestion and (b) preventing new ingestion of lead

For the Housing Environment:

Identifying hazards (a) prior to lead ingestion or (b) after lead ingestion

Major Program Directions:

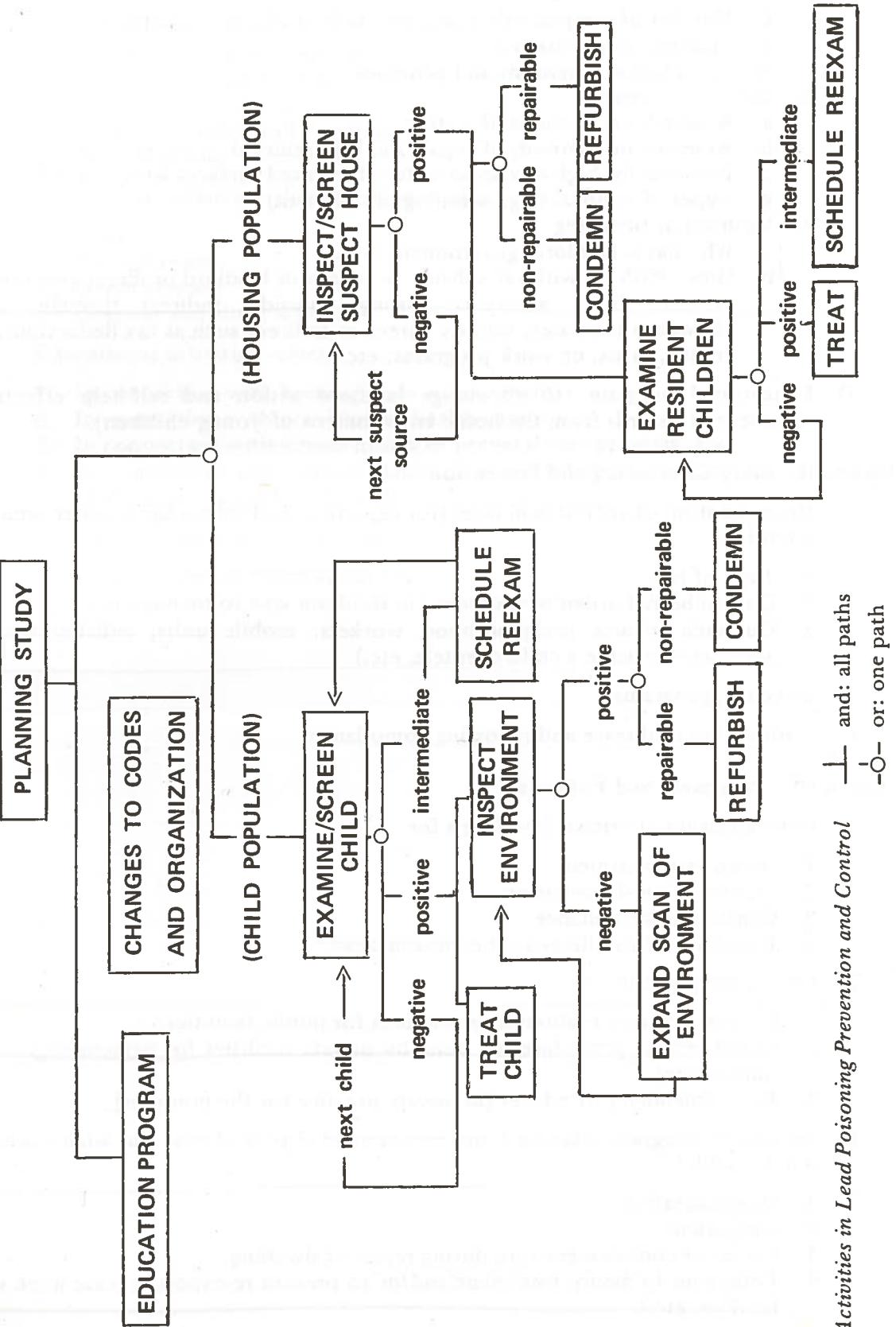
- I. Removal of the hazard from the environment
- II. Early casefinding and prevention
- III. Remedial care of children with lead poisoning and follow-up for vulnerable children
- IV. Health education.

These four program approaches are complementary and within each a variety of techniques and different levels of effort are possible. For convenience, the principal program options are grouped for general description under these four heads, although there are obvious overlaps in the purposes served by the various types of activity.

Program possibilities are summarized below.

Option I: Removal of Lead Hazards from Environment

- A. Inspection prior to health complaints, or following complain
- B. Inspection program (including follow-up and enforcements)
 - 1. Test procedures (public or private)
 - 2. Research for better testing methods
 - 3. Permit requirements or not
 - 4. Tenant information based on inspection
 - 5. Complaints and enforcement methods
- C. Remedial measures



Activities in Lead Poisoning Prevention and Control

1. Paint content restrictions
2. Housing code requirements
 - a. Lead test methods
 - b. Number of samples taken and selection of places to sample
 - c. "Excess" lead criterion
 - d. Compliance provisions and penalties
3. Method of repairs
 - a. Research on methods of testing
 - b. Research on methods of repair and lead removal
 - c. Removal for high-risk areas or for all painted surfaces with tested lead
 - d. Types of removal (e.g., scraping of old paint)
4. Methods of financing
 - a. Who pays: landlord, government, tenant
 - b. How: With or without subsidy to tenant or landlord or direct government project; direct assistance through subsidy; indirect through rental allowance increases; subsidy direct or indirect such as tax deductions, tax credits, loans, or work programs; etc.
- D. Educational program (to encourage landlord action and self-help efforts to remove lead hazards from the home environment of young children).

Option II: Early Casefinding and Prevention

- A. Determination of critical lead level (for reporting, and follow-up or other remedial attention)
 1. Type of test
 2. Critical body burden of lead
 3. Outreach efforts (neighborhood workers, mobile units, collaboration of agencies that serve a child clientele, etc.)
- B. Screening programs
- C. Notification of disease and reporting compliance

Option III: Treatment and Follow-up

- A. Determination of critical lead levels for—
 1. Immediate treatment
 2. Further clinical evaluation
 3. Continuing surveillance
 4. Reporting of dwellings to the housing agency
- B. Evaluation procedures
 1. Standard disease evaluation procedures for public facilities
 2. Reimbursable procedures (if done by private facilities for persons eligible for public care)
 3. Recommended procedures (in private practice for the nonpoor)
- C. Treatment program (standard and recommended procedures, and what costs are reimbursable)
 1. Hospitalization
 2. Outpatient
 3. Foster or convalescent care during repair of dwelling
 4. Follow-up to assure treatment and/or to prevent re-exposure (case work with families, etc.)

Option IV: Health Education on Lead Hazards and on Methods of Correction

- A. Educational activities—for whom:
 1. Parents and caretakers
 2. Landlords and rental agents
 3. Providers of health service
 4. Community workers
 5. Vendors of paint and varnishes
 6. General public
- B. Educational activities—through what media:
 1. Press
 2. TV and radio
 3. Direct contact
 4. Hospital and clinic seminars
- C. Educational activities—when:
 1. In connection with housing code enforcement
 2. In connection with provision of health care
 3. In connection with school health or prenatal care program, etc.
 4. In connection with rentals (landlord responsibility to give notice to tenants about any existing lead hazard)
- D. Educational Activities—what type:
 1. Study of ghetto communication
 2. Development of new methods of communication

Bibliography

1. Specter, Michael J. and Guinee, Vincent F.: Epidemiology of Lead Poisoning in New York City—1970. Presented at the APHA 98th Annual Meeting, Houston, Texas (October 26, 1970).
2. Jacobziner, H.: Lead Poisoning in Childhood: Epidemiology, Manifestations and Prevention. *Clinical Pediatrics*, 5:277-286 (May 1966).
3. Jacobziner, H.: Epidemiology of Lead Poisoning. *Bulletin of National Clearinghouse for Poison Control Centers, Public Health Service*, pp. 7-8 (March-April 1965).
4. Mellins, R. B. and Jenkins, C. D.: Epidemiology and Psychological Study of Lead Poisoning in Children. *JAMA*, 158:15-20 (May 1955).
5. Lin-Fu, Jane S.: Childhood Lead Poisoning—An Eradicable Disease. *Children*, 17:2-9 (January-February 1970).
6. The Surgeon General's Policy Statement on Medical Aspects of Childhood Lead Poisoning: Department of Health, Education, and Welfare; Public Health Service (November 1970).
7. Greengard, Joseph: Lead Poisoning in Childhood: Signs, Symptoms, Current Therapy, Clinical Expressions. *Clinical Pediatrics*, 5:269-276 (May 1966).
8. Bradley, J. E.; Powell, A. E.; Niermann, W.; McGrady, K. R.; Kaplan, E.: The Incidence of Abnormal Blood Levels of Lead in a Metropolitan Pediatric Clinic with Observation on Value of Coproporphyrinuria as Screening Test. *Journal of Pediatrics*, 49:1 (July 1956).
9. Griggs, R. C.; Sunshine, I.; Newill, V. A.; Newton, B. W.; Buchanan, S.; Rasch, C. A.: Environmental Factors in Childhood Lead Poisoning. *JAMA*, 187: 703 (March 1964).
10. Byers, R. K.: Lead Poisoning—Review of the Literature and Report on 45 Cases. *Pediatrics*, 23: 585-603 (March 1959).
11. Christian, Joseph R.; Celewycz, Bohdan S.; Andelman, Samuel L.: A Three-Year Study of Lead Poisoning in Chicago. *AJPH*, 54:1241-1245 (August 1964).
12. Chisolm, J. J., Jr., and Harrison, H. E.: The Exposure of Children to Lead. *Pediatrics*, 18:943 (1956).
13. American Academy of Pediatrics Subcommittee on Accidental Poisoning: Prevention, Diagnosis, and Treatment of Lead Poisoning in Childhood. *Pediatrics*, 44:291-297 (August 1969).
14. Chisolm, J. J., Jr.: Lead Poisoning. *Scientific American*, 224:15-23 (February 1971).
15. Moncrieff, A. A.; Koumides, O. P.; Clayton, B. E.; Patrick, A. D.; Renwick, A. G. C.; and Roberts, G. E.: Lead Poisoning in Children. *Archives of Diseases of Childhood*, 39:1-13 (1964).
16. Greenberg, M.; Jacobziner, H.; McLaughlin, M. C.; Fuerst, H. T.; and Pellitteri, O.: A Study of Pica in Relation to Lead Poisoning. *Pediatrics*, 22:756 (October 1958).
17. Chisolm, J. J., Jr., and Kaplan, Eugene: Lead Poisoning in Childhood—Comprehensive Management and Prevention. *Journal of Pediatrics*, 73:942-950 (December 1968).
18. Feigin, R. D.; Shannon, D. C.; Reynolds, S. L.; and Shapiro, L. W.: Lead Poisoning in Children. *Clinical Pediatrics*, 4:38-45 (January 1965).
19. Smith, Hugo D.: Pediatric Lead Poisoning. *Archives of Environmental Health*, 8:68-73 (February 1964).
20. Nelson, W. E. (editor): *Textbook of Pediatrics*. Philadelphia: W. B. Saunders Company, 1966; pp. 70-76.
21. Lourie, R. S.; Layman, E. M.; Millican, F. K.: Why Children Eat Things That Are Not Food. *Children*, 10:143-146 (July-August 1963).
22. Personal communication with Henrietta Sachs, M.D.
23. Millican, F. K.; Layman, E. M.; Lourie, R. S.; Takahashi, L. Y.; Dublin, C. C.: The Prevalence of Ingestion and Mouthing of Nonedible Substance by Children. *Clinical Proceedings of the Children's Hospital of the District of Columbia* (August 1962).
24. Schroeder, H. and Tipton, I.: The Human Body Burden of Lead. *Archives of Environmental Health*, 17:965-978 (December 1968).
25. Kehoe, R. A.: Metabolism of Lead in Man in Health and Disease (The Harben Lectures 1960). *Journal of the Royal Institute of Public Health and Hygiene*, 24:81, 101, 129, 177 (August 1961).
26. Berman, E.: The Biochemistry of Lead: Review of the Body Distribution and Methods of Lead Determination. *Clinical Pediatrics*, 5:287-291 (May 1966).
27. King, B. G.: Maximum Daily Intake of Lead Without Accumulation of Excessive Body Lead-Burden (unpublished data).
28. Goldsmith, J. R. and Hexter, A. C.: Respiratory Exposure to Lead: Epidemiological and Experimental Dose-Response Relationships. *Science*, 158:132-134 (October 1967).
29. Parry, W. H.: Lead in Drinking Water. *Lancet*, p. 1207-1208 (December 2, 1967).
30. Patterson, C. C.: Contaminated and Natural Lead Environments of Man. *Archives of Environmental Health*, 11:344-360 (September 1965).
31. Goldwater, L. J. and Hoover, A. W.: An International Study of "Normal" Levels of Lead in Blood and Urine. *Archives of Environmental Health*, 15: 60-63 (July 1967).
32. Kehoe, R. A.: Normal Metabolism of Lead. *Archives of Environmental Health*, 8:232-235 (February 1964).
33. Chisolm, J. J., Jr.: Chronic Lead Intoxication in Children. *Developmental Medicine and Childhood Neurology*, 7:529-536 (1965).
34. Haley, T. J.: Chronic Lead Intoxication From Environmental Contamination: Myth or Fact? *Archives of Environmental Health*, 12:781-785 (June 1966).
35. Kehoe, R. A.; Cholak, J.; Hubbard, D. M.; Bomback, K.; and McNary, R. R.: Experimental Studies on Lead Absorption and Excretion and Their Relation to the Diagnosis and Treatment of Lead Poisoning. *Journal of Industrial Hygiene and Toxicology*, 25:71 (1943).
36. Freeman, R.: Chronic Lead Poisoning in Children: A Review of 90 Children Diagnosed in Sydney, 1948-1967. Part 2: Clinical Features and Investigations. *The Medical Journal of Australia*, pp. 648-651 (March 28, 1970).
37. Langham, W. H.: Radioisotope Absorption and Methods of Elimination: Relative Significance of Portals of Entry. In: Cattellott, R. S. and Snyder, L. A. (editors): *Symposium on Radioisotopes in the Biosphere*. Minneapolis: University of Minnesota Press, 1960.

38. Stopps, G. J.: Symposium on Air Quality Criteria—Lead. *Journal of Occupational Medicine*, 10:550-559 (September 1968).

39. Robinson, M. J.; Karpinski, F. E.; and Briege, H.: The Concentration of Lead in Plasma, Whole Blood and Erythrocytes of Infants and Children. *Pediatrics*, 21:793 (1958).

40. Hamilton, A.: *Industrial Toxicology*. New York: Paul B. Hoeber, Inc., 1934; pp. 43-44.

41. Cantarow, A. and Trumper, M.: *Lead Poisoning*. Baltimore: The Williams and Wilkins Company, 1944; pp. 97, 98, 143.

42. Report of Subcommittee on Accidental Poisoning; Statement on Diagnosis and Treatment of Lead Poisoning in Childhood. *Pediatrics*, 27:676-680 (April 1961).

43. Biehusen, F. C. and Pulaski, E. J.: Lead Poisoning After Ingestion of a Foreign Body Retained in the Stomach. *NEJM*, 254:1179 (1956).

44. *Control of Lead Poisoning in Children*. Pre-publication Draft, U. S. Department of Health, Education, and Welfare, Public Health Service, Bureau of Community Environmental Management; pp. I-2-I-3 (December 1970).

45. Bell, W. B.: Influence of Lead on Normal and Abnormal Cell-Growth. *Lancet*, 1:267-276 (1924).

46. Hardy, H. L.: What is the Status of Knowledge of the Toxic Effects of Lead on Identifiable Groups in the Population? *Clinical Pharmacology and Therapeutics*, 7:713-722 (November-December 1966).

47. Byers, R. K.: Round Table Discussion. *Proceedings of the Lead Hygiene Conference*. Chicago: Lead Industries Association, 1958; p. 73.

48. Wiener, Gerald: Varying Psychological Sequelae of Lead Ingestion in Children. *Public Health Reports*, 85:19-24 (January 1970).

49. Berg, J. M. and Zappella, M.: Lead Poisoning in Childhood with Particular Reference to Pica and Mental Sequelae. *Journal of Mental Deficiency Research*, 8:44-53 (June 1964).

50. Greengard, J.; Zollar, L.; and Sharifi, M.: Medical Progress in the Prevention of Childhood Lead Intoxication. *Illinois Medical Journal*, pp. 615-618, 640 (May 1968).

51. Chisolm, J. J., Jr.: personal communication.

52. Greengard, J.; Adams, B.; and Berman, E.: Acute Lead Encephalopathy in Young Children. *Journal of Pediatrics*, 66:707 (April 1965).

53. Greengard, J.; Rowley, W.; Elam, H.; and Perlstein, M.: Lead Encephalopathy in Children. *New England Journal of Medicine*, 264:1027 (May 1961).

54. Chisolm, J. J., Jr.: The Use of Chelating Agents in the Treatment of Acute and Chronic Lead Intoxication in Childhood. *Journal of Pediatrics*, 73:1 (July 1968).

55. Coffin, R.; Phillips, J. L.; Staples, W. I.; and Spector, S.: Treatment of Lead Encephalopathy in Children. *Journal of Pediatrics*, 69:198 (1966).

56. Kubota, J.; Lazar, V. A.; and Losee, F.: Copper, Zinc, Cadmium, and Lead in Human Blood from 19 Locations in the United States. *Archives of Environmental Health*, 16:788-793 (June 1968).

57. Woods, G. E. and Walters, R. M.: Lead Poisoning in Mentally Subnormal Children. *Lancet*, p. 592 (September 29, 1964).

58. Lin-Fu, J.: personal communication.

59. Sachs, H. K.; Blanksma, L. A.; Murray, E. F.; O'Connell, M. J.: Ambulatory Treatment of Lead Poisoning: Report of 1155 Cases. *Pediatrics*, 46:389-396 (September 1970).

60. 1970 Census of Population, First Count Tape—District of Columbia Totals. Table 18.

61. Hearings on S.3216 and H.R. 19172 before the Subcommittee on Health of the U. S. Senate Committee on Labor and Public Welfare of the 91st Congress, Second Session. p. 208.

62. Sachs, H. K.: Preliminary data on Chicago (unpublished).

63. Lead Poisoning Screening, June 2-8, 1970 (District of Columbia). D. C. Health Services Administration, Department of Human Resources.

64. Kaplan, E. and McDonald, J. M.: The Blood Lead Value as an Aid in the Diagnosis of Lead Poisoning. *Journal of Pharmacology and Experimental Therapy*, 63:17 (1938).

65. Perlstein, M. and Attala, R.: Neurologic Sequelae of Plumbism in Children. *Clinical Pediatrics*, 5:292-298 (May 1966).

66. Byers, R. K. and Lord, E. E.: Late Effects of Lead Poisoning on Mental Development. *American Journal of Diseases of Children*, 66:471-493 (November 1943). Based on conversation by study staff with District Training School, Dec. 1970.

67. Based on conversation by study staff with District Training School, Dec. 1970.

68. Harris, J. W.: *The Red Cell: Production, Metabolism, Destruction; Normal and Abnormal*. Cambridge, Mass.: Harvard University Press, 1965; p. 27.

69. deBruin, A. and Hoolboom, H.: Early Signs of Lead-Exposure, A Comparative Study of Laboratory Tests. *British Journal of Industrial Medicine*, 24:203-212 (1967).

70. Hernberg, S. and Nikkanen, J.: Enzyme Inhibition by Lead Under Normal Urban Conditions. *Lancet*, p. 63 (January 10, 1970).

71. Lane, R. E.: Health Control in Inorganic Lead Industries. *Archives of Environmental Health*, 8:55-62 (February 1964).

72. Henderson, D. A.: The Aetiology of Chronic Nephritis in Queensland. *Medical Journal of Australia*, pp. 337-386 (1958).

Guidelines for Sampling the Housing Environment

1. *Guidelines for Sampling* — The conditions which require sampling of the environment for lead are:

a. Any reported case with or without evidence of pica referred to the Housing Division, Department of Economic Development by a health service.

b. Requests for testing of painted surfaces for lead when there is reported pica or peeling paint but does not involve a reported case of lead poisoning. A complete investigation is usually not necessary.

c. If a child with lead poisoning spends a great deal of time at other locations where there is a reasonable opportunity for the child to be exposed to a lead source.

d. When a child undergoing treatment for lead poisoning moves to a new address, samples should be taken at the *previous address* and the *present address*.

2. *Number and Location of Sampling* — Normally, at least two paint samples shall be collected from each room. These two samples shall include the window sill and door or door frame. In addition, samples shall also be collected from other highly suspected areas including surfaces with loose paint. The preferred locations to be sampled should include surfaces which have been chewed or eaten, suspected of having been chewed or eaten, or areas which are peeling or flaking. All interior sampling should generally be limited to that part of the surface which may eventually be required to be removed as listed in the Specifications for Removal of Lead Paint. Other samples might also include paint from suspected locations such as crib railings, playpen railings, porches, porch railings, fences, outbuildings, etc.

3. *Amount and Method of Sampling* — The paint shall be scraped to the base surface and at least a teaspoonful, if possible, collected from each location. Samples of plaster, wallpaper, and similar surfacing material shall be taken *only* if they have been painted and a scraped sample cannot be obtained. Collect one sample from each specific location; do not make a composite from different locations. Use a double fold self-sealing flap envelope for collecting the sample. Record all samples collected at a premises on one form. Samples should be sent directly to the Bureau of Laboratories, Department of Public Health, 300 Indiana Ave., N.W., for analysis.

4. *Not too Obvious Sources of Lead* — It may not always be readily evident that a child has chewed or picked on painted surfaces or has eaten flakes or chips of paint which may have fallen from the ceiling or other locations and frequently the parent may not be aware or admit that the child has had a history of pica. It is for these reasons that samples are collected from surfaces where the signs of chewing may not be obvious. In these cases, however, a more thorough interview and investigation of the environment will be required to determine in which room and at what other premises the child may have spent a great deal of time. If you believe that there may be other sources, the guide of possible sources of lead should be used in collecting other types of samples. If sampling of water for lead analysis is necessary, a polyethylene bottle should be used. Before collection the bottle should be rinsed out well with distilled water. The sample should be taken from the first slug of water from the tap in the morning. This technique will insure the highest probable lead content since the sample has been in contact with the pipes overnight.

5. *Interpretation of Laboratory Results* — Where the laboratory results reveal that the paint samples contain more than one percent (1%) lead by weight, the paint is leaded and therefore unsafe, in accordance with the United States of America Standards Institute Z66.101964—USASI Standard Specifications to Minimize Hazards to Children from Residual Surface Coating Materials. A copy of the lab report will be sent to the Accident Prevention Division. The Neighborhood Health Clinic or district nursing office, and the Attending Physician through the hospital clinic will be notified of the results by the Accident Prevention Division.

Suggested Procedure for the Issuance of Orders for Removal of Lead Paint

1. Issuance of an order is not an automatic action based solely on a request for an investigation. An order is issued to the responsible person when the following conditions exist: (see unusual circumstances below)

a. A case is reported by a physician regardless of the blood lead level (based on physician's judgement of clinical and laboratory findings), and

b. The child exhibits pica either observed directly by first hand evidence, or reported by parent or physician, and

c. There is any source of lead in the environment (exceeding 1% by weight) as determined by laboratory analysis.

2. The issuance of orders in unusual circumstances is governed by the following situations:

a. If the lead poisoning victim has moved to a new address, an official order on the previous address is not issued. However, if the *previous address* contains a lead hazard the owner should be requested by letter to remove it before the dwelling is rented to a family with young children.

b. If the child continues to exhibit pica at the *new address* and the child is exposed to lead paint an order is issued to the responsible person of the *new address*.

c. If a child spends a great deal of time at another location that contains a lead hazard in the environment an order is not usually issued, except in cases where there is a baby sitting business, nursery, etc. In cases where enforcement is not indicated, a letter should be sent to the owner advising him of the hazard and precautions to be taken.

d. The standard order should not be issued to another city agency on property which they own or manage. Such cases should be discussed with the agency for appropriate action.

3. The order directs the responsible person to eliminate the hazardous condition within 10 days and in emergency situations, within 3 days. The order will also state that the owner (or occupant) should not repaint until an inspection is made by the Housing Division, Department of Economic Development to determine if the hazard was satisfactorily eliminated. Copies of "Specifications For the Removal of Lead Paint and Safety Standards to be Used When Removing Lead Paint from Dwellings and Facilities" should be sent with the order. When the order is issued to the owner, a separate standard letter should be mailed to the occupant advising that a hazard exists and to do his part by not allowing the child to chew on painted surfaces and to sweep up chips and flakes of paint. Three (3) copies of the order and of the letter to the occupant should be sent to the Accident Prevention Division for distribution to appropriate persons.

Suggested Specifications For the Removal of Lead Paint

The following specifications are intended as a guide for the responsible person to whom the order is issued, to eliminate the hazard of lead paint. The sanitarian making the follow-up compliance inspection should use these specifications in determining whether compliance with the order has been achieved.

Lead paint creating a health hazard to children shall be removed as follows:

The person to whom the order is issued should:

Step A	Refer to the Order for the room or location where lead paint must be removed, and
Step B	Find the specific areas at such locations on the list below, then
Step C	Remove all layers of lead paint to the length, width, and height as indicated below:

1. Cracked, chipped, blistered, peeling or other loose lead paint shall be completely removed wherever found, holes and cracks in walls must be repaired.
2. Windows, sills and frames below 5 foot level—*complete removal*.

3. Doors and frames below 5 foot level — *removal 4 inches back on hinge and latch edges of door, complete removal from frame*
4. Handrails — *complete removal.*
5. Spindles (Balusters) — *removal on surfaces adjacent to walking areas.*
6. Stair treads — *removal 4 inches back from lip on top of tread and from lip to riser on bottom side.*
7. Other chewable surfaces below 5 foot level — *removal 4 inches back from edge.*

TIGHT LEAD PAINT surfaces which do not require removal include: walls in good condition without broken areas, baseboards, skirtboards on stairways, step risers, and any other surface below the 5 foot level not presenting a chewable surface.

IN LIEU OF REMOVAL of the lead paint as specified above, the surfaces shall be covered with an approved durable material. Such materials may include metal, hard fiber board, tile, plastic board, or any other material approved by the Department of Economic Development.. *Repainting a surface with a non-leaded paint without the complete removal of the existing lead paint shall not be deemed to be satisfactory.*

If the responsible party intends to repaint, it shall not be applied until the areas from which the paint is removed has been inspected and approved in writing. After notification of satisfactory compliance by the environmental health inspector, any paint safe for interior use (1% lead or less) may be applied.

Suggested Follow-up Compliance Inspection

A follow-up investigation shall be made by the inspector immediately after the date set for compliance on the order to determine if compliance has been effected in accordance with the order. If the surface has been repainted without prior inspection and it is not possible to determine if the surfaces were scraped satisfactorily, then additional samples to the base surface shall be collected to determine compliance with the order; it will normally not be necessary to sample the new paint. The person to whom the order is issued shall be notified in writing of non-compliance or satisfactory compliance. A copy of this notice should be sent to the Accident Prevention Division, who will notify the Neighborhood Health Center or appropriate district nursing office and attending physician through the hospital or clinic.

RESEARCH NEEDED

Major Areas:

- Lead hazard - (Under what conditions, and at which levels is lead damaging to brain cells; at which levels do psychological changes occur?)
- Tenant response to information on environmental hazard
- Motivation of families:

To screen children for lead burden
To receive medical care
To control lead ingestion

- Extent of recurrence of lead poisoning.
- Environmental clean up methods, and incentives for landlords
- New and improved screening and diagnostic methods:

For the child
For the housing environment

- New and improved methods of treatment.

